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ORIGINAL ARTICLES

SOME FACTORS IN THE DIAGNOSIS OF CARDIAC CONDITIONS.*

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It is my belief, based upon a fairly large clinical experience, that exaggerated importance has been attached to cardiac murmurs in the diagnosis of cardiac disease. I am sure this is true with apical systolic murmurs. I believe the distinction should be made at once between apical systolic murmurs before forty, and after forty. At the latter age they may mean early myocardial degeneration. Presystolic or diastolic murmurs favor organic disease. In the absence of murmurs serious organic lesions have been overlooked. Had the evidence for or against cardiac disease been obtained the real diagnosis, independent of the presence or absence of murmurs, would have appeared. Just consider for a moment what a variety of heart conditions, clinically, are associated with murmurs:

Malformations of the heart.
Malpositions.
Open foramen ovale.
Acute or chronic valvular defects.
Calcified deposits upon the valves.
Atheroma of the arch.
Syphilis of the arch.
Fibrinous and adhesive pericarditis.
Pleuro-pericarditis.
Hypertrophy of the ventricle with relative mitral leakage.
Dilatation of the ventricle.
Anaemic murmurs.
Cardio-respiratory murmurs.
Relative incompetence from high pressure.

In contrast to the above causes of murmurs I would call your attention to the following clinical facts: With extreme heart weakness no murmur may be heard; in angina pectoris there may be no heart enlargement, no murmurs, and a low blood pressure; in mitral disease the murmurs at the time of examination may be absent, or heard only in some unaccustomed place, as in the left lateral position, or only after exercise; systolic basal murmurs—the earliest manifestation of arterio-sclerosis—may sometimes be heard only with the patient on the back, with the breath held in deflation; the diastolic murmur of aortic regurgitation at times appears not at the base but at the apex—upon placing its origin depends the diagnosis—while the diastolic murmur of syphilitic aortitis with regurgitation is so low pitched, in the early stage, as to require frequent and repeated examinations to determine its presence. Suspicion of its existence from the known etiology, leads to this search. This point is worthy of emphasis. *Suspicion of some cardiac condition,* based on other factors than murmurs, will often times lead to the discovery of murmurs.

The question of the interpretation of murmurs has become acute because so many men with apical systolic murmurs have been rejected in the draft. I have had the opportunity to examine a certain number of these; in none have I been able to discover evidence of organic disease. The size of the heart was normal by accurate X-ray measurements; the cardiogram tracings have been normal. One striking feature in all of the cases was the low position of the heart—the ptotic heart. So difficult is it to discover the important murmur—so many times the murmur is absent at examination—that I believe it is time to emphasize other factors in the diagnosis of heart conditions. Relegate most murmurs, at least the apical systolic ones, to an inferior position in diagnosis, not a superior one.

Cardiac disease arises, in the main, through (1) infections which damage the endocardium, the pericardium, or the myorcardium. Syphilis, while more selective, may affect the first part of the aorta with secondary valve retraction. (2) Degenerative changes of unknown or toxic origin by damaging vessels, especially the coronary arteries, produce weakened heart muscle. In general, it may be said heart disease arises

*Read before the Providence Medical Association, December 3, 1917

either from infections or degeneration. Of the infectious causes of heart disease in the young, chorea, rheumatic fever, and tonsillitis are pre-eminent. Anyone of these makes probable organic mitral or aortic disease, with or without pericarditis, acute or chronic, with or without acute or chronic glomerulo nephritis. Pure aortic regurgitation, in early adult life, presupposes a syphilitic aortitis with or without aneurysm, possibly with early tabes. Myocardial infection from syphilis undoubtedly occurs—it is less easy to recognize clinically. Arterio-sclerosis produces its chief cardiac damage by lessening the blood supply through narrowed coronaries—hence a myocardial weakness. High pressure, from whatever cause, may be associated with heart symptoms. These symptoms, in my experience, depend upon the integrity of the coronary circulation, and the ability, therefore, of the heart to hypertrophy. Given high pressure (200 systolic) in the young adult, from chronic glomerulo nephritis and there may be no heart symptoms, even in the presence of heart enlargement to two or three times the normal, until weakened by the terminal uraemia or anaemia, the heart muscle dilates. Clinically these cases, waterlogged, resemble mitral regurgitation with failure of compensation. Usually the aortic second plus will make the diagnosis clear. On the other hand cardiac symptoms may appear early in other pressure cases, when coronary circulation is impaired and heart muscle nourishment interfered with. In other words, before you have heart disease you must have had a cause for heart disease. Search out the cause—infectious, syphilitic, nephritic or degenerative. Practically all organic mitral disease is infectious. Aortic disease may be infectious, probably is if combined with organic mitral. If pure, it is probably syphilitic. Degenerative changes are suggested by weakened heart muscle, anginal attacks, increasing blood pressure. Roughly speaking, before the age of twenty exclude infections. After twenty, look out for the nephritic heart and for early syphilis. At forty, or thereabouts, syphilis is common in its heart manifestations. After forty, syphilis and early arterio-sclerosis should be considered. Given good blood supply to the heart one would expect, if damage to the heart, vessels or kidneys threw extra work on the heart, there would be increase in its size. To determine whether the heart is or is not enlarged I believe one of the most important factors in the diagnosis of heart disease. This determination is not always easy when the enlargement has been chiefly left ventricular. It enlarges backward, and escapes recognition by the usual method of percussion. If left ventricular hypertrophy is suggested by the history or symptomatology, turn the patient on the left side and percuss the left ventricle, now near the chest wall, and palpate in the fifth, sixth or seventh interspace for pulsation, noting the force. If heaving and felt in the sixth, undoubted ventricular hypertrophy is present. Do not be misled by the low position of the ptotic heart. Your percussion area as usually obtained may show a heart of normal size. Palpation and percussion in the left lateral position may show considerable left ventricle enlargement. One is wrong, the other is right, and the diagnosis and further study of the heart proceed from this observation.

I was led to adopt this left lateral percussion by comparing the size of the heart as ordinarily estimated by percussion with the real size at autopsy. To have normal percussion limits and a heart enlarged to two or three times the normal size at post mortem, led me to investigate, and I discovered the mistake occurred only where the enlargement was chiefly left ventricular, in which case posterior position of the enlarged left ventricle prevented its recognition by anterior percussion.

If X-ray plates and cardiogram tracings were possible in all patients many unrecognized cardiac cases would be discovered, based upon this failure to recognize left ventricular hypertrophy by ordinary percussion. Right sided hypertrophy is usually easily recognized by percussion, as is left sided hypertrophy when associated with dilatation. It is the recognition of left ventricle hypertrophy before the ventricle dilates that is important. To find out the cause as early as possible, whether nephritis, syphilis, or arterio-sclerosis, makes diagnosis more accurate and treatment more effective. I would rather have the left border percussion line with the patient in the left lateral position than any other in a patient over forty. The two next in importance are the cardio-hepatic angle, and the width of sub-sternal dulness. The width of sub-sternal dulness at the second and third ribs usually is between 5 and 6 cm. In high pressure, arterio-sclerosis, especially in syphilitic aortitis, this dulness may reach 7 or 8 cm. Glands may mis-

lead; occasionally the dilated left auricle in mitral stenosis is encountered in this percussion.

I would again emphasize these two points—(1) Ascertain an etiology for the suspected heart lesion. Frequent tonsillar attacks or rheumatic fever make valvular damage a probability. I would study most carefully any apical systolic murmur in a patient with a chronic tonsillar history, especially if the tonsils were hypertrophied or merely clipped, not excised. (2) Estimate the size of the heart, thereby judging whether any increased work had been forced upon it by the suspected damage. With no enlargement the lesion is either recent or of slight extent. This applies to valve conditions essentially.

A lesion of any moment will usually manifest itself either by dyspnoea on exertion, pain on exertion, palpitation, pounding or irregular heart action. Many of these symptoms, or all of them, may occur in the individual case. To attach importance to a systolic apical murmur with no known etiology obtained for organic valve condition, with no demonstrable heart enlargement by accurate percussion, checked up by X-ray heart measurements seem to me to attach too much value to one bit of evidence and neglect other and more important facts.

I seldom examine any patient where a systolic murmur is not heard, after exertion, with the patient in the left lateral position. This often is loud and of wide transmission. It is probably due to relative mitral leakage, not organic. If these systolic murmurs are so easy to call forth by exercise, and change of position, and arise from so many causes they must have a varying degree of importance. At present this importance is in direct ratio to the experience of the examiner. When a systolic murmur is heard at the apex the next statement made is "transmitted or not transmitted to the axilla." Organic murmurs are, as a rule, more widely transmitted in mitral cases toward the left axilla. It is true, clinically, they are more often of recognized pitch, or have a musical quality or rough character, rather than a mere whiff or souffle. But, since mitral regurgitation is usually associated with some degree of mitral stenosis, usually other facts to support the organic character of the supposed mitral lesion may be obtained, as, for example, a sharp first sound, a reduplicated first sound, a presystolic or a diastolic murmur. It may require exercise or change of position to obtain this evidence of mitral stenosis, but once obtained the real value of the systolic apical murmur appears, and a definite diagnosis of mitral disease, organic, may be made. Usually the right ventricle or left auricle will show enlargement if the lesion is extensive. Usually the pulmonic second will be accentuated.

To approach the value of murmurs from still another angle may be of interest. Given an etiology for cardiac damage—take syphilis, for example—without knowing what murmurs syphilis may produce, you would overlook the earliest cases of syphilitic aortitis—the most important to recognize. A systolic murmur at the base of the heart in a man of forty years, who had syphilis at twenty or twenty-five, may be the earliest and only sign of aortitis. If by percussion or X-ray you find a beginning arch dilatation, or obtain a history of angina showing coronary involvement in the process, or are fortunate enough to find the diastolic murmur showing beginning value retraction, your diagnosis is made at once—due, first, to the syphilitic history; second, to the systolic murmur. Without these facts or knowledge of syphilitic aortitis this patient might have been assured that no serious condition was present, so frequent are basal systolic murmurs. Or, again, given syphilis in the history, unless especial care was taken to eliminate early aortitis by most prolonged search for the systolic basal murmur, the real lesion would be overlooked and the heart pronounced sound. Here, again, I am emphasizing the value of etiology and a knowledge of pathology, which makes hunting for murmurs of known importance essential. These murmurs are of importance because they have a reason for their existence; they occur in the proper place, after the proper interval after infection—they have a right there from etiology and pathology.

Besides etiology and cardiac enlargement, a third and very important fact in the diagnosis of heart conditions is the limitation or non-limitation of the field of cardiac response. What symptoms has the patient had pointing to a weak heart? Dyspnoea on exertion is the most striking and constant one. Pain on exertion is perhaps the most important one. Sensations such as those produced by extra systoles are misleading and require tracings for their proper interpretation.

Oftentimes a careful history will show that for years, especially in chronic valve infections, there has been less and less ability to work, walk

or sleep except on higher pillows, without symptoms of cardiac discomfort. This fact of the progressive character of the limitation points at once to increasing mechanical valve defects or weakening heart muscle. Contrast this with the sudden onset, after some unusual exertion, of cardiac pain or dyspnoea in a man of forty-five robust and rugged, as he thought, up to the moment of his discomfort. Recognizing the possible syphilitic cause in this case at once leads to the inquiry of the time of infection—it will usually antedate the cardiac symptoms by twelve to fifteen years. Cardiac symptoms usually precede death by two years. Not to recognize late syphilitic aortitis is bad enough; not to recognize early aortitis is worse. How important it is to recognize by history alone slight limitations of activity, from dyspnoea or mild angina, the earliest symptoms of the early stage of the senile heart, before hypertrophy has been obtained. The entire plan of treatment aims to favor hypertrophy by limitation of exertion and use of digitalis. Nothing may be found on examination to point directly to the heart, but after you have seen these cases over a series of years, you learn what will happen if the recognition of the condition is postponed.

You may examine a patient and find no heart enlargement, no increase of pressure, no murmurs, and pronounce the heart sound. Death may occur in twenty-four hours from cardiac disease—serious—easily recognized at the time of examination. Had you asked yourself the question: Is this patient of the infectious age, the syphilitic age, or the degenerative age? and questioned accordingly you would undoubtedly have found two factors—(1) cardiac pain on exertion; (2) limitation of the field of cardiac response. Either of these two would have enabled you to make the diagnosis: angina pectoris, arterio-sclerosis, with beginning senile heart. The earliest sign of serious cardiac mischief in this type of cases may be obtainable only through the history. Angina may be the first symptom before you get any enlargement, or even the relative mitral of a weakening myocardium. Recently upon the almost forgotten history of pain on exertion six years before, in a man fifty-five or six years of age, I made the diagnosis of angina. There was no enlargement; blood pressure 130 systolic, 100 diastolic; a seven foot X-ray plate showed no abnormality; the cardiogram tracing was pronounced normal by an expert; and yet within two weeks after the examination I had opportunity to see the occluded left coronary, the occluding right coronary, with fibrosis of the myocardium—the patient dying in an attack of angina. Six years ago the diagnosis should have been made—the end result is not capable of treatment.

It has not been my purpose to touch upon arythmia, tachycardia, fibrillation, flutter, block or those especial technical cardiac conditions. I could not if I would—they must be left in the hands of a trained expert. The diagnosis of many cardiac conditions is impossible without cardiogram or polygraph tracings. But when I see such simple methods as a search for a cause, sound cardiac size estimation, and the estimation of the field of cardiac response neglected, and over-emphasis placed, by the average physician, upon murmurs, I cannot but feel that something is wrong. I have disregarded hundreds of apical systolic murmurs after study of the case—there was no cause for them in the history; there was no evidence from cardiac size that they increased the work of the heart; there was no effect upon the activity of the patient by their presence. In my experience they occur in nervous people with low pressure, oftentimes with ptotic hearts.

MEASLES.

D. L. Richardson, M. D.,
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Synonyms: Morbilli, Rubeola. French: Rougeole. German: Masern. In this country "measles" is the name most widely used and is by far the best. If a scientific name is used, "morbilli," the Latin name for the disease, is to be preferred since the term "rubeola" may be easily confused with "rubella," the proper name of the disease poorly called "German measles." Furthermore, the term "rubeolé" is the French name for rubella. "German measles" is a name that should be dropped.

It is stated by medical historians that measles had its origin in the countries bordering on the Red Sea, about the tenth century, A. D. The first accurate description of the disease was made by Rhazes, a distinguished Arabian physician. While knowing little or nothing about the history of medicine, I venture to say that measles, as well as all infectious diseases, had

their origin at a much earlier period of the history of man. In the earliest times, to be sure, they may have been very limited in distribution because of lack of social or commercial intercourse. Undoubtedly, however, measles appeared as an epidemic in the tenth century and fell under the eyes of a keen observer who was able to set it aside as a new disease—not an altogether new disease, for all the Arabian physicians believed it to be a variety of smallpox, bilious smallpox, it was called by Avicenna. It was not until Sydenham studied an epidemic from 1670 to 1674 that the symptoms were fully described and the disease determined as an infectious entity and not a manifestation of smallpox. Up to Sydenham's day, and even later, measles and scarlet fever were considered the same disease, and again he made a valuable contribution to medicine by accurately describing scarlet fever, to which little has been added to the present day.

Measles is to be found in all parts of the world. Age, sex, season, or race will not protect against this, the most highly transmissible of all the infectious diseases, the one which very few human beings escape. It would seem that the first three or six months of life are singularly free from attack, altho the disease may possibly be contracted *in utero*. Children between one and ten years of age are most commonly attacked. This is explained by the highly transmissible character of the disease and the universal susceptibility to it. It is usually contracted at the first exposure. Adults are as susceptible as children, provided they are not protected by a previous attack. Panum states that in the Faroe Islands, in 1846, not one person who had escaped in the epidemic of 1791 escaped the disease. The interval between these epidemics was over fifty years. Geographical position cutting off human travel may keep communities free from the disease for long periods of time.

Etiology and Epidemiology.

The exact cause of measles remains still unknown. A bacillus has been recovered by Pielicke, Carron, and Barbier. A similar short, slender bacillus has been found in the blood by Borini, who was able to produce with it, in small animals, a disease resembling measles. Lesage has described a micrococcus which he succeeded in isolating from the blood and nasal mucus and with which he claimed to produce in rabbits a disease resembling measles. But none of the above work has been corroborated.

Something quite definite has been learned about the body fluids which contain the measles virus and how long these fluids are infectious. The most recent work has been done by Goldberger and Anderson of the United States Public Health Service, and Hektoen of Chicago. It is interesting to know that Horne of Edinburgh, in 1758, claimed to have inoculated twelve children with blood from measles lesions which was rubbed into excoriated surfaces. He failed to inoculate children intra-nasally with nasal secretions. In 1822, Speranza successfully inoculated measles; about 1854, Bufalini reported successful inoculations of his own and of three other Italian investigators. In 1842, Katona inoculated 1,122 individuals with blood plus the contents of miliary vesicles, and claimed ninety-eight per cent. of this number developed measles. He states that fever developed at the end of seven days. Mayr successfully inoculated measles, in 1848, by placing nasal mucus in the noses of two children. The period of incubation was eight or nine days. He was unsuccessful when using blood.

In all the above inoculations, the resulting disease was mild in character. Many of the results have been much questioned, in the past, as other observers were unsuccessful. Viewed, however, in the light of recent investigations, it would seem that at least part of the results mentioned above can be relied upon. During 1911, Goldberger and Anderson made a great contribution to our knowledge of the virus of measles by utilizing monkeys, which are susceptible both naturally and artificially to the disease. Since then Hektoen and Eggers of Chicago, Nicolle and Conseil of Paris, Lucas, and Prizer, have successfully inoculated monkeys.

In experimental cases, about fifty per cent. of the animals show definite symptoms. The period of incubation is not less than five days, but it is variable. A rise in temperature is constant. Some cases show catarrhal symptoms, while others do not. This is true of the eruption. Inoculation protects against a second successful inoculation. The measles virus was carried through six monkey generations. Finding that they were susceptible to inoculation experiments, they exposed animals to other animals who were suffering from the disease and transmitted it in this way. In this connection, they mention an

instance reported by Chavigny in which a monkey caught the disease from its keeper, with whom it had been in close contact. They have shown that the nasal and buccal secretions contain the virus, but the scales of desquamation do not. They have shown that the blood loses its infectivity, thirty-six to forty-eight hours after the appearance of the eruption. The secretions collected during the first forty-eight hours after the appearance of the eruption contained the virus, and the infectivity of these secretions was much diminished or lost with the approach of convalescence. The virus will mostly pass through a Berkefeldt and is, therefore, a filterable virus. It resisted dessication for twenty-five and one-half hours; is killed by heat of 55.° C. exposed for fifteen minutes; resists freezing for twenty-five hours, and retained infectivity after twenty-four hours at 15.° C.

Measles is the most highly transmissible of all the infectious diseases. There is almost universal susceptibility. Exceeding few persons who live to middle age escape it. Of the number who escape, some undoubtedly suffered from it in some unrecognizable form. It would be strange if all cases of measles developed the rash upon which we largely depend for diagnosis. If it always conformed to the text-book picture, measles would differ from all other infectious diseases. In a large family, there may be one or more members who apparently escape the disease in its usual form who have some transient catarrhal symptoms which are undoubtedly due to measles. I have seen two or three cases of measles without a rash.

The disease is endemic in large cities, taking an epidemic form every two or three years. Smaller communities may be entirely free from it for several years until introduced by some case from outside the town. During this period of freedom there have grown up a considerable number of susceptible children and the disease continues until few escape. The classic example, which I have already referred to, is the Faroe Islands, which were free from the disease from 1781 to 1846. It was then introduced by a cabinet-maker, who contracted it in Copenhagen. Six thousand people out of a population of 7,782 took measles before it had spent its force. Every old adult who had not had the disease in 1781 contracted it in 1846.

The infective agent of measles probably does not live long outside the human body. Most observers agree on this point. From the work of Goldberger and Anderson, it would appear to live longer than commonly supposed. However, the danger of a physician or other exposed person carrying it to others in a community is very slight indeed; but under hospital conditions or in institutions into which measles has been introduced, infection by nurses, physicians, and others who pass between the sick and the well, is certainly carried on the hands, and so forth, unless rigid precautions are taken. The French and English hospital statistics bear this out. Kerr of Edinburgh believes that he has seen it carried by nurses. At the Providence City Hospital, I am convinced that it has been carried by nurses, by physicians, or by both.

This leads up to the mooted question of air or droplet infection. The English authorities, with varying degrees of certainty, believe that droplet infection does occur, Kerr of Edinburgh and Rundle of Liverpool dissenting. Our own experience has been as follows: Since the opening of the hospital up to the end of 1912, we have treated 207 cases of measles in our isolation wards. In 1910, from 59 cases treated in these wards, only one case developed. 1911, from 26 cases, 11 cases developed. These eleven developed between October 30 and December 12, and from a single case which was admitted during the incubation period. It will be noted that for one and one-half years previous to the date October 30, only one cross infection developed. During 1912, from 121 cases, 12 cases developed; these were during the early part of the year and followed on the heels of the previous outbreak. No case developed after March 15. A careful study of the circumstances leads me to a strong belief that air dissemination played little or no part. It was noted that a patient in the opposite end of the ward was as likely to be the first to develop the disease as the patient across the hall. We have to deal with two possibilities: either someone has been careless in technic, or else the virus is hard to remove from the hands when in a very fresh state. I am convinced that measles is a contact disease, for the above reasons.

The period of infectivity is pretty definite. The latter part of the incubation period is undoubtedly infectious, reaching its height during the latter part of the pre-eruptive and the first

part of the eruptive stage, and ceases not far from four days after the beginning of the rash. The Health Department of New York city quarantines measles for only six days after the beginning of the eruption.

The period of incubation of measles is more definite than for almost any other infectious disease, small pox only approaching it. From nine to ten days are required from the exposure to the first symptoms, and thirteen to fourteen days from such exposure to the appearance of the rash. In animal experiments, this period is shorter, just as it is in such experiments with all other infectious diseases because the virus is usually introduced directly into the blood.

Fever and slight indisposition are the very first symptoms of measles, the catarrhal symptoms coming on later and being of varying intensity and length. I have some twenty-four charts of cases of measles which have contracted the disease in the hospital and on whom bi-daily temperatures were being taken for the respective diseases from which they were suffering at the time the new infection took place. It is to be noted that temperature was usually the first sign of the disease except in a few instances when a rise in pulse preceded any temperature. In one instance a rise in pulse was the only sign of the disease until the day of the rash, when the temperature rose. In a few instances the rise in pulse, which is never out of proportion to the rise in temperature, did not follow for a day or two after the development of temperature.

The average length of the prodromal period was four and one-third days. It was five days in ten cases; four days in nine cases; three days in four cases; and two days in one case. It will be noted that the rash usually appeared on the fourth or fifth days of the diseases.

In general three types of temperatures were observed. The most frequent is that given by the text-books, and appears in twelve of the twenty-four cases: namely, a fall within 24 to 36 hours after onset of rash, this remission lasting usually about 24-36 hours and again rising to the fastigium. The fastigium usually coincided with the height of the rash. It may precede the height of the rash, but rarely follows this point.

The fastigial temperatures were as follows:

107.0	1 case
105.0	1 "
104.8	1 "
104.0	9 "
103.8	2 "
103.6	1 "
103.0	5 "
102.6	1 "
101.0	3 "

These temperatures were rectal, with four possible exceptions. Another point which I wish to emphasize is the early drop of temperature after the appearance of the rash. To look at the chart, it is seen that the major portion of the illness precedes the eruption and this pre-eruptive period, during which the disease is not usually recognized, is, unfortunately, the time when it is most infectious. I do not wish to be understood to say that temperature is necessarily the very first symptom, but I believe that it is the first to be elicited from children who are not able to express a slight feeling of malaise. From the histories of adult cases, the incubation period is not always free from slight symptoms. Such an example may be a 22-year-old girl who complained of epigastral pain and vertigo eleven days before the appearance of the rash.

The intensity and duration of catarrhal symptoms preceding the eruption were rather variable. In a few cases, these symptoms were exceedingly slight, so that the rash was about the first thing noted.

There occurs rather constantly a leucocytosis beginning during the last two or three days of the incubation period and lasting out the period of invasion, usually falling to normal about the time of the development of the eruption. The leucocytosis is of the polymorphonuclear variety. This agrees fairly well with the experiments on monkeys conducted by Hektoen and Eggers, except that there is a relatively high lymphocyte count, which is true of the monkey in normal health.

I do not propose to discuss the general symptoms, course, complications, or sequelae of measles, but cannot close without calling to your attention unrealized facts about the mortality of the disease. Kerr of Edinburgh in a recent article publishes some mortality statistics for measles and other diseases in different countries from 1887 to 1908, viz.:

DEATH RATES PER MILLION LIVING (1887-1908).

	United Kingdom	France	Prussia	Austria	Belgium	Holland	U.S.A.
Measles.....	354	810	260	382	457	258	101
Scarlatina....	135	171	275	511	158	31	104
Diphtheria....	199	864	750	788	315	127	87

It is evident from this table that nearly as many children die of measles as from diphtheria and many more than from scarlet fever. In Aberdeen, for nineteen years, the case fatality was 3.33 per cent. For the first year of life it was 13.9 per cent.; second year, 10.0 per cent.; third year, 3.4 per cent.; fourth year, 1.6 per cent.; and thereafter less than 1.0 per cent. This is in a city which has a good Health Department and where diseases are well reported. In many other communities, the case rate is much higher. In the report from which the above figures are taken Dr. Wilson says that within ten years (previous to 1904) measles had caused more deaths than any other zymotic disease and even more than all other zymotic diseases put together, excluding whooping cough.

Hospital mortality is considerably higher than the foregoing figures. In all the London fever hospitals for the past two years, it has been about 13 per cent. for all cases and ages. At times it reaches 25 per cent.

It is interesting to note that the total and the case fatality of measles and whooping cough is about the same.

LEAD POISONING, A CAUSE OF OBSCURE DISEASE.

By W. LOUIS CHAPMAN, M. D.,
Providence, R. I.

It is the purpose of this brief communication to call the attention of the medical and dental professions to the probability of the greater frequency of chronic lead poisoning than we are accustomed to believe. It was in the study of obscure cases of gastro-intestinal disease that the writer had made examinations of the urine for lead and obtained a sufficient number of positive findings to cause him to believe that, with certain symptoms, no study of the case was complete without eliminating the possibility of lead as a causative factor. The usual classic symptoms of lead poisoning are well known. The lead line, wrist drop and colica pictonum, together with a history of occupational exposure, readily suggest lead, but the chronic form in which none of these symptoms occur, or at least occur in mild or obscure form, is probably very often overlooked. It is easy to diagnose diseased conditions when evinced by marked physical signs. It is not as easy when the signs are suggestive rather than positive or when they are seemingly subjective rather than objective. This is true of cardiac, gastric and in fact of almost any form of disease.

That it is of the greatest importance to diagnose disease in its incipience is also self-evident, as is also the fact that many symptoms considered as subjective, if observed for a time, are later shown to be caused by pathological conditions and would have been so considered had one's studies been sufficiently thorough. I believe this to be true of lead and that in the past a great many cases have been unrecognized because of failure to make the necessary tests in cases showing articular and muscular pains, malaise and asthenia, gingivytis with loss of teeth often without suppuration, indigestion and irregularity of the bowels and a considerable variety of objective and subjective gastro-intestinal symptoms. Then again a great many cases have escaped us because we did not think of the possibility of lead. This is not at all surprising because the state of our knowledge is such that we naturally think of lead toxemia as an occupational neurosis or disease and hardly one to be found among the nobility and the gentry. Again, the process of the examination of the urine for lead, which is the diagnostic proof, is somewhat complicated and decidedly expensive and one to which recourse would usually be had only in exceptional cases. From the brief experience of the writer, however, it will be seen that we must examine the urine for lead more frequently if we wish to cure our cases and relieve them of their troublesome symptoms.

No doubt every practitioner recalls cases which he did not help until potassium iodide was administered, and although signs of syphilis were not present, yet he felt that perhaps such a patient had syphilis and the iodide assisted in the cure. It is highly probable that such cases might have been due to lead, for in lead poisoning the relief afforded by eliminants is immediate and marked.

In the State Board of Health Journal of Rhode Island for January, 1917, Mr. Gage,

the state chemist, suggested the importance of examinations for lead, and it is due to the State Laboratory that such examinations may be made in cases where patients cannot afford a chemist's fee. My thanks are due the State Laboratory for the examination of the urine in several of my cases. Owing to the fact that K I increases the output of lead in the urine, it may be suggested that it should be given for a week before the sample is submitted, so that subsequent examinations need not be made. This is particularly important where there is but little lead in the system, and it must be noted that cases showing a trace or under .5 mg. may show a much larger amount after taking the iodide.

As yet it is not known how little lead may be ingested and show symptoms, or how much lead may be taken and not cause symptoms. Here as in other diseases the patient may show susceptibility or idiosyncrasy, or the delicacy of the perceptions may fortunately indicate poisoning where it would not show in a less finely organized nervous system. This is also true of malaria in the South, death having occurred without any particular complaint; the same thing has been noticed in cases of beri beri among the fishermen of Labrador.

It is possible that the stippling of the erythrocytes characteristic of lead poisoning may have been mistaken for malaria in some cases, and it is important to note that quinine, in the writer's opinion, is of value in cases of lead poisoning.

.5 mg. is to be considered the danger point of lead in the urine. Any person whose urine shows that amount or over should be observed and treated. How long a time is required to completely free and eliminate the metal is not known. Nor do we know just how long a person should stay under treatment.

With the enormous increase in the consumption of tinned foods there is an added danger, because of the acid nature of some of the contents. Trench warfare has contributed to our knowledge of lead disease from tinned foods and possibly because of some fermented liquors containing lead.

In the etiology of gingyvitis, recession of the gums and dental caries lead must be considered as a very important causative factor. On a number of occasions the writer has suggested the possibility of the syphilitic origin of cases of pyorrhea alveolaris where the disease was widespread and showed several fistulous openings in the alveolar process, for the reason that such cases were often markedly helped by K. I. He now urges the importance of a study for lead in such cases, and believes that his views as to their luetic origin must be modified and that systematic eliminative treatment will be followed by immediate and marked improvement in the dental condition. Cases 1 and 3 which follow illustrate this fact. I have under my care at the hospital at the present time a patient with marked lead gingyvitis with distinct line on the gums who has not worked in lead for nine years. The ipecac treatment of gingyval amebiasis is notoriously unsuccessful and it is quite probable that many of these cases are caused by lead.

We do not know very much about the pathological chemistry of lead in the body. The following important questions await solution:

a. What is the point of saturation of the tissues,—that at which lead is eliminated in the urine?

b. What amount of lead is necessary in the average individual to occasion symptoms?

c. At what time may the individual be declared free from lead?

f. What is the lethal dose in chronic poisoning, and how long and in what quantities will dilute and attenuated solutions of lead cause encephalitis, wrist drop and nerve disintegration?

Case 1. A physician consulted me for obscure gastro-intestinal symptoms. Had pains in various parts of the abdomen like colic. Bowels were irregular. He was tired and run down all the time and felt that something was holding him back. Could not seem to get up the speed he wished and did not know why. Had been rapidly losing his teeth from non-suppurative disease. Had a metallic taste in the mouth in the morning. Weak in the legs and rheumatic pains in the legs and wrists. Examination of the urine showed 2 mg. of lead per liter, and after a week's treatment with K. I., 20 grs. per day, the urine showed 5 mg. of lead per liter. The tap water showed 2.78 mg. lead per liter—more than the first examination of the urine showed. Continuing the use of K. I. and using spring water, showed an immediate and progressive improvement in this case. The abdominal pains gradually subsided, the legs more strong, the bowels

more regular; he did not tire as easily; the loose teeth became more firm and his dentist expressed amazement at the rapid and marked improvement in his gums.

CASE 2. A woman 65 years old. Has had diabetes for many years, with mitral chronic endocarditis, general asthenia and nervousness. Feels dizzy most of the time and has been seen by a neurologist, who diagnoses her case as cerebral arteriosclerosis. She has occasional attacks of colonic stasis and twice has had to have manual removal of rectal scybalae. Occasionally her abdomen swells up and she has intermittent pains all over the abdomen. Of particular interest were sharp pains in both legs over the tibialis anticus and in the right wrist. There is considerable loss of power in both hands. Her urine showed 19 mg. of lead per liter and the tap water .3 mg. per liter. Her improvement under treatment was immediate and marked, and she was much pleased at the cessation of pain in her legs and wrists. She, too, has gingyvitis with complete denudation of her right lower central incisor exposing the apex.

CASE 3. A woman 55. She is a chair invalid or rather spends her time on a couch. Has not walked for six years. Advanced rheumatoid arthritis. Her husband died at age of 57 after an illness of two weeks from nephritis. This patient has deeply congested gums, but very little pyorrhea. It has been suggested that her arthritis was caused by her teeth, but on account of her condition she could not visit a radiologist for a search for apical abscesses. Her urine showed 7.33 mg. of lead per liter.

CASE 4. A woman of 79. She has acute mitral and aortic endocarditis with much dizziness and some dyspnea. Occasional pains over the appendix and gall bladder so severe as to require morphine by the mouth. The symptoms seemed to indicate appendicitis and cholecystitis with lithiasis, but hypodermics were not necessary at any time. Rest in bed, nitroglycerine and other drugs helped her, but it was not until K. I. was given as a chance prescription, a not unusual thing in practise, that she began to improve. Examination of the urine showed 12. mg. lead per liter.

CASE 5. A woman of 46. Has had two operations for intestinal obstruction due to bands and adhesions. Her circulation is poor, she has occasional dyspnea, cold feet and chronic indigestion with occasional attacks of extreme constipation with much abdominal pain. It is quite a study of treatment and diet to keep her in a state of fair health. The urine shows 2.5 mg. lead, which undoubtedly accounts for some of her symptoms.

CASE 6. A woman of 42. Has tachycardia with dyspnea. Heart is usually 120 to 136, with only rarely a soft systolic mitral murmur. X-ray of the chest shows a small heart with old peribronchial enlargement. She rarely has colds and has no signs whatever of an active tuberculosis. Her urine after a week's treatment with K. I. shows 2.5 mg. of lead, and eliminative treatment has reduced her heart to 80 and she has much better circulation than she has had for a long time.

In addition to these cases I have four others showing small amounts of lead, but as they have not had any eliminative treatment, the analysis is not a fair estimate of the amount of poisoning now going on.

These cases have all been assembled during the past 30 days and represent only my own practise and mostly patients that have consulted me for the relief of gastro-intestinal conditions. A practitioner with a very large practise told me recently that he had never had a case of lead poisoning to treat.

There is no doubt but that in some cases the lead poisoning had been going on for years. There are in this city hundreds and perhaps thousands of houses, both old and new, piped with lead, and it is to be believed that there are many persons going from one doctor to another seeking relief of symptoms caused by lead. I believe that the experience of other physicians will be the same as my own if this possible cause of disease is borne in mind and the urine examined for lead in all obscure cases of this type.

CONCLUSIONS.

1. Chronic lead poisoning is undoubtedly very common in this city.

2. It is the cause of many cases of obscure and atypical gartre-enterological and neurological disturbances which are difficult of diagnosis and resistant to treatment. It causes many symptoms before gross pathology is apparent. The lead line appears later.

3. A promising field for study and research

is opened whereby our present incomplete knowledge may be enriched.

4. The pathological chemistry of the blood and tissues is as yet entirely unexplored and we do not know what effect chronic poisoning may have upon cellular pathology and the etiology of new growths.

5. We seek information as to tolerance, selective affinity, susceptibility, treatment, idiosyncrasy and symptomatic diagnosis.

6. It may be that in plumbism we have an important etiological factor as yet unappreciated in gout, the arthritides, occupational neuroses and gastro-enterological manifestations without gross pathology as revealed by our present methods of investigation.

7. Chronic plumbism is probably a widespread cause of dental disease causing interstitial and suppurative gingivytis, recession, focal necroses and premature loss of teeth.

8. Potassium iodide and quinine have a helpful effect in these cases as eliminants and antidotes.

9. The examination of the urine for lead is as yet our chief means of diagnosis in this class of cases.

CLINICAL DEPARTMENT

CRISIS OF PNEUMONIA WITH A LONG DROP IN TEMPERATURE.

By CHARLES E. HAWKES, M. D.,
Providence, R. I.

The following case is interesting on account of the extraordinary drop of 9.2° F. in the patient's temperature at the time of her pneumonic crisis.

Mrs. R., a young married woman, had been successfully confined about two weeks before I attended her. January 13, 1917, her physician, who was ill at the time, asked me to respond to a telephone call that he had received from her. She had been feeling well up to the previous afternoon, when she began to have headache, backache, weakness and lameness all over. Now she had a loose-sounding cough, with a temperature of 103.5° and pulse of 144. Her abdomen and pelvis were negative and lochia had practically ceased. On examination, her chest disclosed no adventitious signs. She was apparently suffering from an attack of the "grippe." Next day she seemed better; temperature was 101.6° and pulse 126. Her cough was still loose and she was expectorating. She had slept well and bowels had moved satisfactorily.

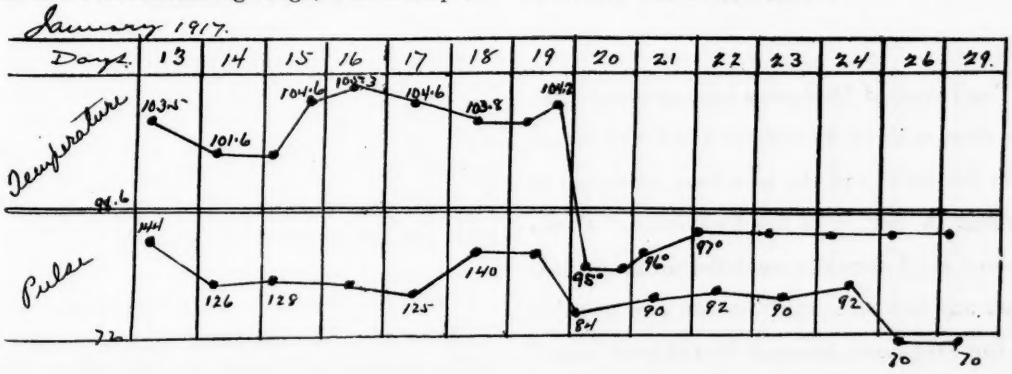
On January 15 she looked a little cyanotic, although her pulse felt fairly strong, at 128 per minute, and no heart murmurs were developing. Temperature remained 101.6°. Her chest condition was the same. Later in the day she felt decidedly chilly and sick, and a nurse was summoned to care for her.

The following day I learned that her temperature had shot up to 104.6° after her slight chill the day before, but had fallen some in the morning. Her color was now normal. A pneumonic spot was developing in her right axilla. Her cough and expectoration remained as before. A dose of castor oil had produced an excellent result. In the evening her temperature rose to 105.3° and later fell to 104.5° after an alcohol bath. The baby had been discontinued at the breast. A second nurse was in attendance at night.

During the evening and night of January 19 her temperature reached 104.2° and pulse 140. She had a tremendous sweat and slept scarcely any.

On the morning of January 20 her temperature had fallen to 95°. It was recorded by both nurses and two different thermometers. Her pulse was 84 and of good quality. Her color showed no cyanosis. She breathed easily and had very little cough, or pain in her lung. Examination of the latter disclosed dry pleuritic friction rubs, near her right breast, and moist rales throughout the upper right lobe.

Her temperature continued 95° for nearly twenty-four hours, and rose to 96° the next day. January 22 it was 97°, where it remained for a week, until I discontinued my visits. Her pulse stayed about 90° until January 26, when it fell to 70°, where it remained. The rest of her convalescence was uneventful.



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Section on Medicine—4th Tuesday in each month, Dr. D. Frank Gray, Chairman; Dr. C. W. Skelton, Secretary and Treasurer

R. I. Ophthalmological and Otological Society—2d Thursday—October, December, February, April and Annual at call of President, Dr. Lewis B. Porter President; Dr. H. C. Messinger, Secretary.

NOTICE

The House of Delegates having voted that the dues shall be $10.00 for 1918, the Treasurer desires to call the members' attention to Article IV Sec. 6 of the By-Laws: "Every Fellow shall annually contribute the Annual dues and the same shall be due and payable to the Treasurer, January first of each year."

EDITORIALS

On the eve of his hurried departure for Halifax, the editor was obliged to turn over the work of bringing out the current issue to his associates on the editorial board, who generously assumed the burden. On his return, he finds the work so well accomplished that the medical public are immeasurably the gainers.

THE PROFESSION AND HOSPITAL.

These are history-making years. Governments, geographical boundaries and nations are

being changed with startling rapidity. In commercial interests new fields are opened and new methods necessary, while social and economic questions that to-day may be solved in a certain way will to-morrow require a new solution. Unwise is the party or government which attempts to solve these problems from their viewpoint alone; equally so the man or body of men who control organizations affecting large numbers if a settlement of difficulties is attempted without considering the rights and interests of all concerned. No man stands so pre-eminently above his fellows that he can give an opinion satisfactory to all, can affect a decision in mooted questions fair to all, and it is for this reason that we rely upon the judgment of a commission which represents all factions. In commerce, in labor, in war, this principle is accepted and followed.

The problem affronting the Rhode Island Hospital this year faces a large deficit, if we correctly understand the appeal of the trustees for greater financial aid, is that either more money must be provided, or the charitable work of the hospital must be curtailed. There are interested in the solution of this equation four factors: the charitable public, the management of the hospital, the medical profession, and beneficiaries of the hospital, and we venture to assert that any decision which rests solely upon the financial side of the question will be inadequate and unsatisfactory. There are other methods which may be employed to make the balance come on the right side of the ledger; there are other people interested in the hospital besides those who contribute financial aid. The medical profession gives to the hospital more than money. It gives its skill, its time and its life blood. It gives what cannot be obtained elsewhere. It gives what is an absolute necessity for the continued existence of the hospital. It knows more about the poor of the city, its needs, and the remedy, than all the social workers combined. It does in aggregate charity more than the hospitals combined. It is vitally interested in all the problems which confront the trustees of the hospital. Why then should not the profession be consulted in this matter? Why should not their opinion and advice be sought? Why should they not have a hearing upon matters in which they are vitally interested?

Occasion has been taken more than once in these columns to urge upon the trustees of the various hospitals that they should elect to membership a physician who could afford information regarding the professional side of hospital work, a phase with which the lay trustee is not supposed to be conversant, or appoint a consulting committee from the staff, or a single member of the staff who could present for their consideration the various problems arising in hospital work and the opinions of the working staff without being considered presumptious. The profession is a unit in its willingness to bear its share of the burden, in eagerness to serve. They deserve, however, some recognition that is not usually accorded to employees.

If those in charge of the various hospitals would invite the profession to unite with them in a study of the problems confronting them, would give weight to their opinions, or a voice in their decisions, they might learn of ways of retrenchment other than cutting salaries; ways of increasing usefulness without increased expense. Inspired by no spirt of fault finding, willing and eager to do their share, the profession is entirely within its right when it asks to be heard.

THE HALIFAX RELIEF.

On Saturday, December 8, 1917, the people of Rhode Island once again were given a demonstration of the spirit of altruistic charity that permeates the body medical of this community. On Thursday the country was shocked by the news of the terrible catastrophe that visited the city of Halifax. On Friday some of the doctors in Providence were asked to volunteer to go to aid the sufferers, and although some of the men did not receive word definitely that they were wanted until after eleven o'clock at night, they were ready to start at eight o'clock Saturday morning. They were told their stay was indefinite. They could not arrange their work and in one case at least, if not in more, their office assistants did not know they were going until after they had gone. This one feature alone is worthy of note. We are somewhat accustomed to seeing the doctor give of his time, energy and skill after he has arranged matters so as not to be at too great a loss financially, but this was an innovation that meant more in a monetary sense perhaps to the men who so generously and promptly dropped everything and went than any other one act within the memory of most of us.

In these horrible war times the demands upon the generosity of the citizens, and that always includes the doctors, is almost innumerable. In the case of the doctor it is doubly hard in that he is expected to donate money as well as his time and skill, while the ordinary layman is asked to give his money only. While this trip was arranged and managed by the local branch of the American Red Cross and any expense involved was paid by them, nevertheless the doctor did in this case again give both his time and his money.

Dr. N. Darrell Harvey of Providence, who was a native of Halifax, was instrumental in the organization of the unit, and after a session with the President of the Rhode Island Chapter of the American Red Cross was able to get the use of five Pullmans and a diner to leave on Saturday morning. Mr. Carl B. Marshall, the Treasurer of the local branch, generously volunteered to go along as the financial man of the party, and proved himself the right man for a very unenviable job. He was able to perform the seemingly impossible feat of inducing the Pullman Company to allow their cars to remain at Halifax to be used by the doctors and nurses as their lodging place while in that city and thus relieve the authorities there of one more burden of providing for them.

The Providence men were joined here by men from Fall River and New Bedford who were to have gone with the Boston Unit. Doctors and nurses were taken on at Woonsocket also, and at Portland a quintet of Social Service Workers from Boston. In all the party totalled 111. When the train pulled into the yard at Halifax they were able to feed and lodge themselves, and were the only Unit to arrive there of which this can be said. They also brought their own nurses and surgical dressings and some foodstuffs. They were not dependent upon anyone in Halifax for anything, and most of the doctors carried their own instruments, in not a few cases some of the men having as high as three and four complete sets of surgical instruments, even to complete laparotomy sets, with all the sterile dressings included. To anyone who knows the amount of detail necessary to collect and arrange these things for an operation when there is plenty of time for their collection and arrangement, it will be little short of marvelous, especially when it is considered that notice was not received by many of them until nearly midnight. It would appear to mean very little sleep. It did mean just that, none for some of them, and yet there was not one sign of complaint or regret from that large number except the displeasure that they were not already on the spot.

It was a splendid tribute to the sterling worth of these good men and women. For the nurses deserve their share of praise as well as the doctors. To them also the sacrifice is one of more than time only. There are very few nurses who have any means of livelihood other than their income derived from nursing. While this is not true of all the doctors, many of them having some slight income, at least, even when they are not practicing their profession at home for pay, but this is hardly true of the nurses.

THE PROBLEM OF CHOREA.

Some of us who have had occasion to observe and to treat any considerable number of patients suffering from chorea hold certain very definite opinions, right or wrong, about it. To begin with, it is necessary to determine with some precision what we mean when we say that a patient has chorea. Turning then to the dictionary we learn that chorea means dancing, and is the word used to denote St. Vitus' dance, which is defined as "a functional nervous disorder usually occurring in youth, characterized by irregular and involuntary action of the muscles of the extremities, face, etc., with general muscular weakness." Reading further we learn that chorea has as synonyms, CHOREA ANGLORUM; CHOREA SANCTI VITI; EPILEPSIA SALTATORIS; ST. JOHN'S DANCE. And if this is not enough to stretch our PIA MATERS, we have only to continue down the page, where we will find standing forth in heavy type a whole Praetorian cohort of choreas, some fifty or more in all. Now, surely, here is some confusion; material, too, as Sir Arthur Quiller Couch might say, for a very pretty little essay upon medical jargon.

Well, then, in the matter of definitions, we have to choose, and so, following tradition, we choose to call that condition, and only that condition, chorea which was so carefully described by Sydenham in 1686, and which is marked by involuntary spontaneous movements, by weakness and want of precision in voluntary movements, and by emotional instability, with, at times, more serious psychical disturbances. If

we keep to our definition, the word will have for us some definiteness of meaning and we shall not call chorea St. Vitus' dance, for it has nothing to do with dancing, St. Vitus' or any other. Why indeed should we continue to talk jargon simply because our forefathers mistakenly did so? Again sticking to our definition, we shall not darken counsel by speaking of, say, paralyzed limbs with athetoid movements as "hemiplegic chorea," nor label as chorea the various habit spasms and tics, for whatever else these are they are not chorea. But, above all, let us not relapse wholly into barbaric speech by committing the solecism of "false chorea,"—twin brother of "pseudo-angina" and the whole uncouth and mongrel brood of words beginning with "pseudo."

We said just now that some of us have certain positive opinions about chorea, of which opinions the most positive is this: Chorea, as defined above, is always the body's reaction to some infection. It may be admitted, and freely admitted, that other factors are at work in this patient and in that, such as emotional strain, developmental instability, fright, poor nutrition and so forth; but we would stoutly defend the proposition,—no infection, no chorea. It is curious that this problem of infection is passed over so lightly in many of our text-books, and those, too, amongst the most recent. Some of them speak of infection from tonsils and adenoids; but of seven which lie before us as we write, not one refers to the role of infected teeth and gums in etiology, prophylaxis or treatment. And yet to any one who has himself seen the rapid and really remarkable improvement in the condition of choreic patients which sometimes follows treatment of these focal infections, such omission seems serious and inexplicable. Knowing, as we do, that endocarditis occurs more frequently in chorea than in acute rheumatism, is it not a reasonable postulate that infection is at the root of the business? And, furthermore, is it not, to say the least of it, a short-sighted policy to put our trust in aspirin, or salicylates, or arsenic, or diet, or anything else, unless we have at the same time discovered, if possible, and removed any source of infection? But not to labor the point further we shall end by claiming that to be a rational working hypothesis which maintains that Sydenham's chorea is the body's reaction to some infection. If such it is, the inference for practice is clear,—remove the infection, and, what is equally important, forestall relapses by preventing reinfection.

RHODE ISLAND MEDICAL SOCIETY.

The records of the December meeting of the State Society are unavoidably delayed owing to the illness of the Secretary, Dr. J. W. Leech. They will appear in the February issue of the JOURNAL.

SOCIETIES

RHODE ISLAND MEDICAL SOCIETY

SECTION IN MEDICINE.

A regular meeting of the "Section in Medicine" was held in the Medical Library on November 27, Dr. D. Frank Gray presiding. The paper of the evening was by Dr. Augustus George Gigger on "Laboratory Technic." Discussion was opened by Dr. W. L. Harris, Dr. Carl D. Sawyer and others.

A meeting of the "Section in Medicine" was held at the Medical Library December 18, 1917, at 8:45 p. m.

Paper: "Vaccine Treatment of Typhoid," by Dr. Henry A. Cooke.

CREIGHTON W. SKELTON, M. D.,
Secretary-Treasurer.

DISTRICT SOCIETIES

PROVIDENCE MEDICAL ASSOCIATION.

The regular monthly meeting of the Providence Medical Association was held at the Medical Library on December 3, 1917. The meeting was called to order by the President, Dr. F. E. Burdick, at 8:35 p. m. There were present at the meeting 74 members and 11 guests. The records of the preceding meeting were read and approved. The application for membership of Dr. John H. Morrissey was read and referred to the Standing Committee.

Dr. Jeannie O. Arnold, Dr. Mary E. Gaffney and Dr. James P. McKenna, having been approved by the Standing Committee, were elected members of the Association.

On recommendation of the Standing Committee, it was voted that members of the Association

in active service of the United States be excused from annual dues beginning January 1, 1918.

In accordance with Article I, Section 6, of the By-Laws, the Standing Committee presented the following nominations for officers and committees for the year 1918:

For President—William F. Flanagan, M. D.

For Vice-President—Harry W. Kimball, M. D.

For Secretary—Charles O. Cooke, M. D.

For Treasurer—Winthrop A. Risk, M. D.

For Member of Standing Committee for five years—Frank E. Burdick, M. D.

For Trustee of the Rhode Island Medical Library Building for one year—Henry C. Hall, M. D.

For Reading Room Committee—George S. Mathews, M. D., Frank T. Fulton, M. D., M. B. Milan, M. D.

For Delegates to the House of Delegates of Rhode Island Medical Society—J. E. Mowry, M. D., Henry J. Hoye, M. D., D. L. Richardson, M. D., P. Williams, M. D., A. D. Rose, M. D., George R. Barden, M. D., W. H. Magill, M. D., E. S. Brackett, M. D., William Hindle, M. D., Albert H. Miller, M. D., Walter G. Sullivan, M. D., Frederic N. Brown, M. D., Harold G. Calder, M. D., Harry C. Messinger, M. D., Robert C. Robinson, M. D.

Dr. Joseph F. Hawkins, business manager of THE RHODE ISLAND MEDICAL JOURNAL, made an earnest plea to the members to aid in securing advertisements for the ensuing year.

The first paper of the evening, entitled "Some Points in the Diagnosis of Heart Disease," was read by Dr. William H. Smith of Boston, Mass. The discussion was opened by Dr. George S. Mathews, who emphasized points brought out by the reader in his paper. The discussion was continued by Dr. J. H. Haberlin, who stated that a murmur heard at the apex of the heart did not necessarily mean heart disease, and also stated that he had under observation four cases of pulmonary stenosis, all over thirty years of age.

The discussion was further continued by Dr. Gray, who emphasized the value of careful history taking, and asked concerning the practical value of the electrocardiogram.

The discussion was closed by Dr. Smith, who stated that the electrocardiogram was of value in cardiac irregularity, heart block and heart weakness.

A rising vote of thanks was given to Dr. Smith for his paper.

The second paper, entitled "Lead Poisoning a Cause of Obscure Disease," was read by Dr. W. L. Chapman. This paper was freely discussed by Drs. Gray, Sundin, Cutts, Farrell, Leonard and Kerney.

The meeting adjourned at 10:20 p. m. A collation was served.

CHARLES O. COOKE, *Secretary.*

HOSPITALS

RHODE ISLAND HOSPITAL.

The annual meeting of the Rhode Island Hospital Staff Association was held at the hospital December 10, 1917, at 8:45 p. m.

Business, election of officers, and selection of time of services.

W. O. RICE, M. D.,
Secretary.

MISCELLANEOUS

Dr. H. E. Blanchard has received a commission as Captain in the M. R. C., U. S. A.

Dr. J. W. Leech has been confined to the City Hospital for several weeks with an attack of diphtheria.

The Memorial Hospital has announced a new schedule of prices as follows: After December 15 the ward rates will be $15 per week, double private room $18 per bed, special nurse's board $7 per week. The following scale for X-ray examinations has been adopted:

Proposed Fee Scale for Roentgen Examinations.

Extremities below hips and shoulders	$ 5.00
Teeth (two positions)	5.00
Simple shoulder	5.00
Difficult shoulder	10.00
Hip	10.00
Chest	10.00
Neck or local spine condition	10.00
Urinary bladder	5.00
Pelvis	10.00
Skull	10.00
Sinuses	10.00
Urinary tract	10.00
Colon (Barium Enema)	10.00
Gall-bladder	10.00
Gastro-intestinal tract (including gall-bladder)	25.00